AMENDMENTS TO THE CLAIMS

This listing of claims will replace all prior versions, and listings, of claims in the application.

Listing of Claims

Claim 1 (Currently Amended): A method of treating diseases associated with endothelial dysfunction which comprises administering a therapeutically effective amount of at least one proteosome inhibitor to an individual in need thereof, wherein the amount is effective to enhance the expression of endothelial nitric oxide synthase (eNOS) and wherein the amount is in a nanomolar range, and wherein the proteosome inhibitor is selected from the group consisting of aclacinomycin A, lactacystin, clastolactacystein, N-carbobenzoxy-L-leucinyl-L-leucinyl-L-leucinal (also referred to as MG132 or zLLL), the boric acid derivative of MG232, N-carbobenzoxy-Leu-Nva-H (also referred to as MG115), N-acetyl-L-leucinyl-L-leucinyl-L-norleucinal (also referred to as LLnL), PS-1 (N-carbobenzoxy-Ile-Glu(OBut)-Ala-Leu-H; SEQ ID NO:1), carbobenzoxy-L-leucinyl-L-leucinyl-L-leucin-vinyl-sulfon, 4-hydroxy-5-iodo-3-nitrophenylacetyl-L-leucinyl-L-leucinyl-L-leucin-vinyl-sulfon (NLVS), pyrazyl-CONH(CHPhe)CONH(CHisobutyl)B(OH)$_2$, benzyloxy-carbonyl(Cbz)-Leu-leuboro-Leu-pinacol-ester, PS-314 (N-pyrazinecarbonyl-L-phenylalanin-L-leucin-boric acid ($C_{19}H_{25}BN_4O_4$)), PS-519 (1R-[1S, 4R, 5S] -1-(1-Hydroxy-2methylpropyl)-4-propyl-6-oxa-2azabicyclo[3.2.0]heptane-3,7-dione ($C_{12}H_{19}NO_4$)), PS-273 (morpholin-CONH-(CH-naphthyl)-CONH-(CH-isobutyl)-B(OH)$_2$ or also referred to as morpholinonaphthylanlanin-Leu-boronate) and its enantiomer, PS-293, PS-296 (8-quinolyl-sulfonyl-CONH-(CH-napthyl)-CONH(-CH-isobutyl)-B(OH)$_2$), PS-303 (NH$_2$(CH-naphthyl)-CONH-(CH-isobutyl)-B(OH)$_2$), PS-321 (morpholin-CONH-(CH-naphthyl)-CONH-(CH-phenylalanin)-B(OH)$_2$), PS-334 (CH_3-NH-(CH-naphthyl-CONH-(CH-Isobutyl)-B(OH)$_2$), PS-325 (2-quinol-CONH-(CH-homo-phenylalanin)-CONH-(CH-isobutyl)-B(OH)$_2$), PS-352 (phenylalanin-CH_2-CH_2-CONH-(CH-isobutyl)l-B(OH)$_2$), PS-383 (pyridyl-CONH-(CHpF-phenylalanin)-CONH-(CH-isobutyl)-B(OH)$_2$), PS-341 (pyrazylcarbonyl-Phe-Leu-boronate); PS-2 (benzyloxycarbonyl)-Leu-Leu-phenylalaninal or Z-LLF-

CHO or Z-Leu-Leu-Phe-CHO), epoxomicin ($C_{28}H_{50}N_4O_7$ or also referred to as Ac(Me)-Ile-Ile-Thr-Leu-EX (SEQ ID NO:5), eponemycin ($C_{20}H_{36}N_2O_5$), Z-Leu-Leu-Leu-al (MG132), CEP1612, dansyl-Phe-Leu-boronate (DFLB), Tyr-Leu_3-VS (SEQ ID NO:2), NIP-Leu-Leu-Asn-VS, Ada-Tyr-Ahx_3-Leu_3-VS (SEQ ID NO:3), Ada-Lys(bio)-Ahx_3-Leu_3-VS (SEQ ID NO:4), dihydroeponemycin, clasto-lactacystin-beta-lacton (omuralid), Ac-Leu-Leu-Nle-al (ALLN), 3,4-dichloroisocoumarin (DCI), 4-(2-aminoethyl)-bezolsulfonylfluorid (pefablock SC), TMC-95-A, gliotoxin, (-)epigallocatechin-3-gallate (EGCG), cathechin-3-gallate, ritonavir, lovastatin, aclacinomicin A (aclarubicin), and cyclosporin, wherein al represents aldehyde, VS represents vinylsulfone, NIP represents 3-nitro-4-hydroxy-5-iodophenylacetate, and bio represents biotin.

Claim 2 (Previously Presented): The method according to claim 1, wherein the diseases associated with endothelial dysfunction are non-insulin related diseases.

Claim 3 (Previously Presented): The method according to claim 1, wherein the endothelial dysfunction is associated with atherosclerosis, coronary sclerosis and coronary artery disease.

Claim 4 (Previously Presented): The method according to claim 1, wherein the endothelial dysfunction is associated with heart failure.

Claim 5 (Previously Presented): The method according to claim 1, wherein the endothelial dysfunction is associated with ischemic diseases selected from the group consisting of peripheral arterial occlusive disease, myocardial infarction and ischemic diseases of organs selected from the group consisting of kidney, spleen, brain, and lung.

Claim 6 (Previously Presented): The method according to claim 1, wherein the proteasome inhibitor is selected from a group consisting of aclacinomycin A, lactacystin, clastolactacystein, N-carbobenzoxy-L-leucinyl-L-leucinyl-L-leucinal (also referred to as MG132 or zLLL), the boric acid derivative of MG232, N-carbobenzoxy-Leu-Nva-H (also referred to as MG115), N-acetyl-L-

leucinyl-L-leucinyl-L-norleucinal (also referred to as LLnL), N-carbobenzoxy-Ile-Glu(OBut)-Ala-Leu-H (also referred to as PS1; SEQ ID NO:1), carbobenzoxy-L-leucinyl-L-leucinyl-L-leucin-vinyl-sulfon, 4-hydroxy-5-iodo-3-nitrophenylacetyl-L-leucinyl-L-leucinyl-L-leucin-vinyl-sulfon (NLVS), pyrazyl-CONH(CHPhe)CONH(CHisobutyl)$B(OH)_2$, and benzyloxy-carbonyl(Cbz)-Leu-leuboro-Leu-pinacol-ester.

Claim 7 (Previously Presented): The method according to claim 1, wherein the proteasome inhibitor is selected from a group consisting of N-pyrazinecarbonyl-L-phenylalanin-L-leucin-boric acid ($C_{19}H_{25}BN_4O_4$) (PS-314); 1R-[1S, 4R, 5S] -1-(1-Hydroxy-2methylpropyl)-4-propyl-6-oxa-2azabicyclo[3.2.0]heptane-3,7-dione ($C_{12}H_{19}NO_4$) (PS-519); PS-273 (morpholin-CONH-(CH-naphthyl)-CONH-(CH-isobutyl)-$B(OH)_2$) and its enantiomer; PS-293; PS-296 (8-quinolyl-sulfonyl-CONH-(CH-napthyl)-CONH(-CH-isobutyl)-$B(OH)_2$); PS-303 (NH_2(CH-naphthyl)-CONH-(CH-isobutyl)-$B(OH)_2$; PS-321 (morpholin-CONH-(CH-naphthyl)-CONH-(CH-phenylalanin)-$B(OH)_2$); PS-334 (CH_3-NH-(CH-naphthyl-CONH-(CH-Isobutyl)-$B(OH)_2$); PS-325 (2-quinol-CONH-(CH-homo-phenylalanin)-CONH-(CH-isobutyl)- $B(OH)_2$; PS-352 (phenylalanin-CH_2-CH_2-CONH-(CH-isobutyl)l-$B(OH)_2$; PS-383 (pyridyl-CONH-(CHpF-phenylalanin)-CONH-(CH-isobutyl)-$B(OH)_2$); PS-341; PS-1 (Z-Ile-Glu(O*t*Bu)-Ala-Leu-CHO [SEQ ID NO:1]); PS-2 [Benzyloxycarbonyl)-Leu-Leu-phenylalaninal or Z-LLF-CHO or Z-Leu-Leu-Phe-CHO); epoxomicin ($C_{28}H_{86}N_4O_7$) and eponemycin ($C_{20}H_{36}N_2O_5$).

Claim 8 (Previously Presented): The method according to claim 1, wherein the proteasome inhibitor is selected from a group consisting of lactacystin and cathechin-3-gallate.

Claim 9 (Previously Presented): The method according to claim 1, wherein the proteasome inhibitor is selected from a group consisting of Z-Leu-Leu-Leu-al (MG132), Z-Ile-Glu(OtBu)-Ala-Leu-al (PS-1) [SEQ ID NO:1], CEP1612, pyrazylcarbonyl-Phe-Leu-boronate (PS-341), dansyl-Phe-Leu-boronate (DFLB), morpholinonaphthylalanin-Leu-boronate (MG273), NIP-Leu_3-vinylsulfone (NLVS), Tyr-Leu_3-VS [SEQ ID NO:2], NIP-Leu-Leu-Asn-VS, Ada-Tyr-Ahx_3-Leu_3-VS [SEQ ID

NO:3], Ada-Lys(bio)-Ahx_3-Leu_3-VS [SEQ ID NO:4], Ac(Me)-Ile-Ile-Thr-Leu-EX (epoxomicin) [SEQ ID NO:5], dihydroeponemycin, lactacystin, clasto-lactacystin-beta-lacton (omuralid), PS-519, Ac-Leu-Leu-Nle-al (ALLN), 3,4-dichloroisocoumarin (DCI), 4-(2-aminoethyl)-bezolsulfonylfluorid (pefablock SC), TMC-95-A, gliotoxin, (-)epigallocatechin-3-gallate (EGCG), ritonavir, lovastatin, aclacinomicin A (aclarubicin), and cyclosporin, wherein Z represents benzyl oxycarbonyl, al represents aldehyde, VS represents vinylsulfone, NIP represents 3-nitro-4-hydroxy-5-iodophenylacetate, and bio represents biotin.

Claims 10-26 (Canceled).

Claim 27 (Previously Presented): The method according to claim 1, wherein the nanomolar range is between 1 and 100 nanomolar.

Claim 28 (Previously Presented): The method according to claim 1, wherein a single administration of the proteosome inhibitor produces a long-term enhancement of the expression of eNOS.

Claim 29 (Previously Presented): The method according to claim 1, wherein the long-term enhancement is for up to ten days.

Claim 30 (Previously Presented): The method according to claim 1, wherein the proteosome inhibitor is MG132.